Journal of Medical and Pharmaceutical Innovation
ISSN : 2347 - 8136

Review Article

Atopic dermatitis – A Homoeopathic Review

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ABSTRACT
Atopic dermatitis is one of the common chronic skin diseases. It's affects infants and children, and persists into childhood. It affects about one fifth of all persons throughout their lifetime, but the prevalence of the disease varies greatly throughout the world. Atopic dermatitis is sometimes referred to as atopic eczema, and for the purpose of this dissertation the term 'dermatitis' and 'eczema' are used synonymously. It is characterized by acute flare ups of eczematous pruritic lesion above dry skin.
Keywords: Homoeopathy, Atopic dermatitis, treatment

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Conflict of Interest: None Declared!

(Received 15 September 2020; Accepted 21 October 2020; Published 22 October2020) ISSN: 2347-8136 ©2020 JMPI

INTRODUCTION

Atopic dermatitis (AD) is one of the common chronic skin diseases. It's affects infants and children, and persists into childhood. It affects about one fifth of all persons throughout their lifetime, but the prevalence of the disease varies greatly throughout the world. Over the last 40 years, the prevalence of atopic dermatitis has risen, perhaps by 2 to 3 folds, in developed or developed nations, and affects 15–20% of children and 1–3% of adults worldwide.

Atopic dermatitis is sometimes referred to as atopic eczema, and for the purpose of this dissertation the term 'dermatitis' and 'eczema' are used synonymously. It is characterized by acute flare ups of eczematous pruritic lesion above dry skin. Atopic dermatitis generally starts in early childhood and may represent the initial step of the so called 'atopic march' which represents the natural history of atopic manifestations, characterized by a typical sequence of atopic diseases in childhood preceding the progress of other allergic disorders later in life.

Fifty percent of all those with atopic dermatitis develop additional allergic symptoms within their first year of life and probably as many as 85% of the patients experience an onset below 5 years of age. Patients generally outgrow the disease in late childhood as approximately 70% of the patients with a disease start during childhood have a spontaneous remission before adolescence. On the other hand, early childhood atopic dermatitis is often the early sign that a child may later develop asthma and/or allergic rhinitis (hay fever).

The sensation of itch is the major symptoms of atopic dermatitis. Symptoms of atopic dermatitis consist of patches of skin that are red or brownish, dry, cracked or scaly skin and itchy skin, mostly at night. In infants, eczema generally appear as tiny bumps on the cheeks, while older children and adults often experience rashes on the knees or elbows (often in the folds of the joints), on the scalp and backs of the hands. Atopic dermatitis poses a significant burden on health care resources and patients' quality of life (mainly because of sleep deprivation due to itchiness, employment loss, time to care and financial costs). As an effect, there has been a heightened interest in the identification of environmental risks and protective factors.

Overall atopic dermatitis has prevalence of 2.3 %. Significant morbidity may outcome with time of work or study, recurrent hospital admission and disturbance of personal and family life.

Epidemiology: Atopic dermatitis affect about one fifth of all persons throughout their lifetime, but the prevalence of the disease varies significantly throughout the world. Over the last

40 years, the incidence of AD has risen, perhaps by 2 to 3 folds, in developed or industrialized nations while remaining low in agricultural nations. The incidence also appears to be higher in the urban areas, compared to rural in developed nations and more common amongst those from higher social classes.

Etiology and pathogenesis: Prevalence of atopic dermatitis is on the increase all over the world and this fact has been especially noted in urban residents. Atopic dermatitis erupts due to a complex interplay between genetic susceptibility genes, environmental and innate immunological factor ultimately leading to barrier damage. This barrier dysfunction plays a significant role in the pathogenesis of atopic dermatitis leading to entry of allergens and microbes. Studies of asthma and atopy have shown that in etiology, the proportion of causative factors is likely to be about 50% environment and 50% genes.

Genetic influence: Genetic influence in atopic dermatitis is well known. It is supported by studies of twins. In two early genetic epidemiologic studies in population based twin samples, the pair wise concordance rate was 0.72-0.86 for monozygotic twins and 0.21-0.23 for dizygotic twins. In children with atopic dermatitis, it is particularly associated with the prevalence of atopic disease in their parent (maternal> paternal). About 27 % of children whose parents are not atopic develop AD versus 38% and 50%, respectively of children with one or two affected parents.

Filaggrin: In 2006, Palmer et al were the first to show, that the two filaggrin (FLG) mutations, R501X and 2282del4, were connected with the development of atopic dermatitis. Numerous studies have replicated this finding, and the mutations are currently the most strongly associated genetic factors known to confer susceptibility to AD in European populations with odds ratios varying between 3.73 and 7.1. No negative or equivocal studies have been reported. 9-10% of the general population carries at least one null mutation in the FLG gene.

Epigenetics: Epigenetic modification means covalent modification of DNA and histones in the cells, which alters the determination of the expression of genes during the cells growth and differentiation. Epigenetic modifications can be heritable, without involving a change in the DNA sequence, or can be due to ecological factors thus disturbing the heritage, onset and progression of atopic dermatitis.

Inflammation of the Skin in atopic dermatitis: Classically, the inflammation in AD is described as a biphasic response with an initial Th2-dominated cytokine profile; for example, high production of IL-4, IL-5 and IL-13 followed by a mixed Th1/Th2 response (e.g., additional production of IL-2 and interferon-c [IFN-c]. IL-22 that originates from the Th22 lymphocytes has been implied in the acute phase of AD as it increases the epidermal growth but down regulates the skin barrier function along with IL-31, which also induces pruritus.

IgE-mediated Allergic Reactivity: The majority of people with atopic dermatitis have a personal or family history of allergic rhinitis or asthma. 80% patients with atopic dermatitis have elevated serum IgE antibodies against airborne or ingested protein antigens while 20% of patients with atopic dermatitis have normal serum IgE and no allergen reactivity and the disease also occurs in a gammaglobulinemic child with no IgE.

Cellular Immune Abnormalities: The accurate role of cellular immune responses was speculated right from the days of noted dermatologist kaposi. He was aware of some immune incompetence in patients with atopic dermatitis because their susceptibility to widespread herpes simplex infection.

Role of infectious agent in atopic dermatitis (Bacteria, viruses, fungi) : In atopic dermatitis patients there is increased affinity to bacterial, viral and fungal skin infections. Staphylococcus aureus is found in over 90% of atopic dermatitis skin lesions. One strategy by which Staphylococcus aureus exacerbates or maintains skin inflammation in atopic dermatitis is by secreting a group of toxins known to act as super antigens, which stimulate marked activation of T cells and macrophages. Most atopic dermatitis patients make specific IgE antibodies directed against the staphylococcal super antigens found on their skin.

The role of food allergy in the development of atopic dermatitis: The ordinary diagnostic approach is to screen children with moderate to severe atopic dermatitis for sensitivity to eggs, dairy products, peanuts, soy, wheat, fish and tree nuts (walnut, cashew, pecan) by using skin prick tests or RAST with supplementary testing for additional suspected foods obtained by the history or given by the patients.

Outside-Inside-Outside" Hypothesis: Whether the defect in cutaneous permeability barrier is a consequence of inflammation or the xerosis and / or permeability barrier abnormality could drive disease activity in atopic dermatitis and other inflammatory dermatoses constitute the “outside-

inside" hypothesis. Three proposed sites for therapeutic intervention in atopic dermatitis. At least 3 pathogenic mechanisms contribute to the pathogenesis of atopic dermatitis and therapies are accordingly aimed at them.

Role of histamine and neuropeptides in atopic dermatitis: Since antihistamines (HI and H2 blockers) do not relieve itching in atopic dermatitis, histamine is not an important mediator for the pruritus of atopic dermatitis. A third histamine receptor (H3) and a fourth histamine receptor (H4) expressed on numerous immune and inflammatory cells may also be responsible for this itching.

Socioeconomics: A higher prevalence of AD has repeatedly been observed among high income family's independent from household size and the number of older siblings., this was seen in physician diagnosed dermatitis and could therefore not be explained only be differences among social classes in respect to reporting and labeling of symptoms.

Maternal factors: It has been observed that atopic disorders are more frequently transmitted to the child by mothers than by fathers possible mechanisms are:

- Suppression of paternal genomic effects.
- Intrauterine programming (a major factor of which is the balance between fetal nutrition and growth rate).
- Immunological sensitization through intrauterine exposure to food and environmental allergens which the mother is subjected to.

Environmental factors : Although many different environmental risk factors have been considered potentially causative for atopic dermatitis, only a few are consistently accepted. For example, there is substantial evidence that our western lifestyle leads to some of the reported increase in eczema occurrence over the past years although this has not pointed to specific environmental risk factors or has translated directly into functional preventive measures.

Diagnostic criteria: Diagnosis of atopic dermatitis is based on a group of signs and symptoms as there are no laboratory "gold standard" for the diagnosis of AD. The definitive diagnosis of AD requires the presence of all three of the following features: Pruritus, typical morphology and distribution, and chronic and chronically relapsing course. In majority of the cases, the diagnosis is quite simply through in routine dermatologist office with the help of signs and symptoms, history, morphology and distribution of skin lesions and associated clinical signs. Hanifin and Rajka's criteria for the diagnosis of atopic dermatitis.

Clinical features of Atopic dermatitis according to age[1]		
Phase	**Age of onset(year)**	**Clinical feature**
Infant	<2	Typically develops after 2nd month of life
		Edematous papules, papulovesicles, and/or evolving plaques with oozing and crusting over the cheeks (centro facial sparing)
		Face and neck are affected in over 90% of the patients, in first 6 months Sparing of diaper area
Childhood	2-12	Lichenified, less exudative lesions
		Flexural eczema is characteristic (antecubital/popliteal fossae)
		Head, especially the periorificial regions, neck, wrists, hands, ankles, and feet are often affected Pronounced and widespread Xerosis
Adolescent Adult[1]	>12	Chronic hand dermatitis (both endogenous and exogenous components)
		Others have facial dermatitis with severe eyelid involvement
		Erythrodermic disease is prominent in those with continuous AD since childhood

Senile AD- is seen in age above 60 years, characterized by Xerosis, lichenified flexural lesions usually are not present

DIFFERENTIAL DIAGNOSIS OF ATOPIC DERMATITIS: The differential diagnosis of atopic dermatitis is closely associated to the age of the patient. It includes other forms of dermatitis, immune deficiencies related with eczematous rashes, infectious diseases and infestations, metabolic diseases, neoplastic diseases and other chronic inflammatory skin conditions.

Management

General management: Treatment has to be directed against all the known factors, but the basic principle is to prevent scratching. Reassurance, explanation and encouragement for child and parents are perhaps more important for this than any other chronic diseases. Causative factors known to increase atopic dermatitis must be reduced, e.g. soap, wool, and extremes of climates.

Food allergy: Food allergy is mainly prevalent in young children with moderate to severe dermatitis. A Danish population based study found that 14.8 % of children suffered from food allergy and of these, 90 % had atopic dermatitis. An undetected food allergy may result in severe allergic reactions in the child, including respiratory symptoms and anaphylaxis, but it may also worsen the atopic dermatitis.

Probiotics: There have been a number of studies dealing with the effect of probiotics on atopic dermatitis. Most studies have shown beneficial effects of the use of supplementation with probiotics in mothers and infants in preventing development and reducing the severity of atopic dermatitis.

Psychosomatic Approaches: A recent study by Chrostowska Plak and colleagues evaluated the association among itching and stress, health associated quality of life (HRQoL) as well as depression in adult patients with AD, and it was shown that patients with symptoms suggesting depression had more increase itching compare with the rest of the patients.

Psychological strain can stimulate escalation in AD activity and sickness representations and coping are highly associated with self rated physical impairment in AD patients. Further, it has been demonstrated that psychological interventions have a positive effect on pruritus in atopic dermatitis.

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